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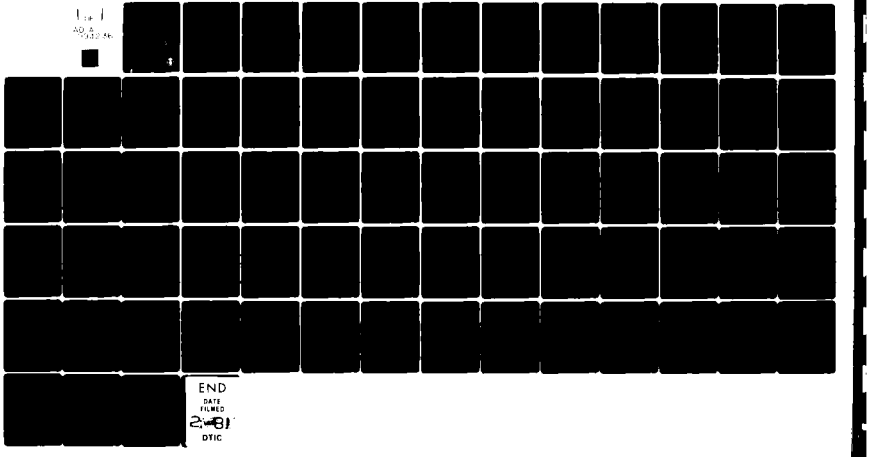

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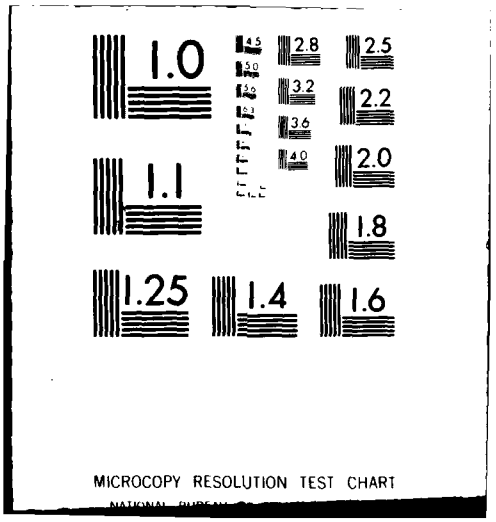
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Technical Report 7906

PROBLEM DEFINITION STUDIES ON POTENTIAL ENVIRONMENTAL POLLUTANTS

VII. PHYSICAL, CHEMICAL, TOXICOLOGICAL, AND BIOLOGICAL PROPERTIES OF DDT AND ITS DERIVATIVES

JUNE 1979

Prepared for the OFFICE of the PROJECT MANAGER
for CHEMICAL DEMILITARIZATION and INSTALLATION RESTORATION

by

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This report is a data base of physical, chemical, toxicological, and biological properties of DDT. Sufficient data are presented to allow a preliminary assessment of the human health impacts and ecological impacts of DDT at sites within the United States where DDT is found. This report is intended to be used with chemical analysis data of DDT concentrations in water, sediments, soils, and biota at particular sites of interest to develop a qualitative assessment of potential hazard.

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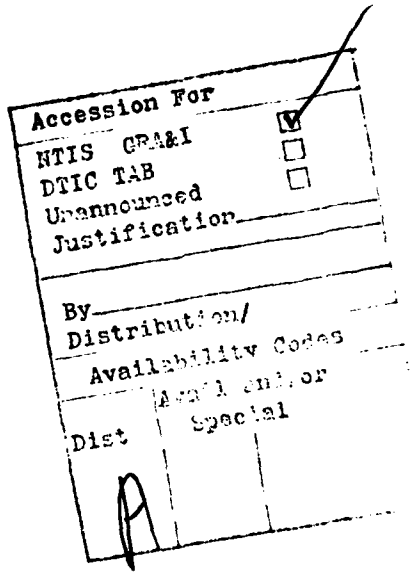

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I. INTRODUCTION

Requirement for Report

The U.S. Army Toxic and Hazardous Materials Agency (USATHAMA), formerly the Office of the Project Manager for Chemical Demilitarization and Installation Restoration, has identified an initial list of substances requiring assessment because of their actual or potential presence in the environment outside the boundaries of Pine Bluff Arsenal (PBA), Arkansas (Table I-1).[1] The U.S. Army Medical Bioengineering Research and Development Laboratory (USAMBRDL) has divided the list into logical units for problem definition studies. Substances used in pyrotechnic devices are treated in two reports.[2,3] Thiodiglycol and elemental phosphorus have been assessed previously in reports by Rosenblatt et al.[4,5] and Dacre and Rosenblatt;[6] a separate report on these substances specific to PBA has been deferred indefinitely. DDT is considered separately here because (a) it is neither military-unique nor installation-unique; (b) there is an overwhelming amount of information available in the published literature; and (c) most pertinent data have been summarized in review articles. The present report deals exclusively with DDT, its isomers and metabolites.

Format

The format of this report departs from that of previous reports in this series[2-5,7,8] because it incorporates both the data base and site-specific

TABLE I-1. POLLUTANTS AT PINE BLUFF ARSENAL[a]

DDT
Thiodiglycol
Phosphorus (white)
Auramine
Benzanthrone
1,4-Di-p-toluidinoanthraquinone
1,4-Diamino-2,3-dihydroanthraquinone
1-Methylaminoanthraquinone

a. As provided in Reference 1.

considerations for a hypothetical installation. There are two reasons for this approach. First, Redstone Arsenal (RSA) also has major DDT contamination, and USATHAMA personnel have indicated that RSA data are as important to their mission as PBA data. Second, contamination surveys and corrective measures were initiated at both PBA and RSA while this report was in preparation. Thus, in view of the continuous output of new data, it appeared neither practical nor useful to analyze site data for either installation.

Instead, a hypothetical site has been created (Section VII) to illustrate the qualitative relationships of DDT levels in water, sediment, and biota to effects of DDT on health and the environment. Quantitative considerations for this site are derived from fragmentary data available for PBA and RSA at the time this study was initiated. This section may be used to estimate the potential ecological effects of DDT waste disposal relative to past known or postulated declines in wildlife populations as well as to the lower DDT concentrations in soil and water resulting from cleanup operations. An important caveat must be given here. Concentrations of DDT in soil, sediment, water, and biota, and the toxic effects predicted therefrom, have been derived using concentration factors, i.e., the ratio of DDT in sediment to DDT in water, DDT in biota to DDT in water, etc. To do so is strictly valid only if these concentration factors represent true equilibrium or steady state values. In very few cases are data sufficient to make such a distinction, and for this reason, soil, sediment, water, or food chain concentrations predicted to lead to a particular toxic effect may be in error by an order of magnitude.

Scope

This report is ecologically oriented. Mammalian toxicology and human health effects of DDT have been exhaustively reviewed in a 1979 document of the World Health Organization (WHO).[9] Some representative data are included in the present report, but investigators concerned with human health aspects of DDT (and the tradeoff between health benefits and hazards) should refer to the WHO text.

The volume of data on environmental effects of DDT has obliged USAMBRDL to exercise considerable and arbitrary selectivity in choice of material to review. For the most part, data relevant to the environments of south central Arkansas and northern Alabama have been collected. The ecological literature has been surveyed systematically through mid-1976 and selectively thereafter. Because of the availability of many definitive reviews, efforts concentrated on surveying the literature of the last ten years, and few references published prior to 1970 were retrieved. In the case of aquatic organisms, a search was conducted not only for DDT, but also for the seven isomers and metabolites detected in the soil of PBA--p,p'-, o,p'-, and m,p'-DDT; p,p'- and o,p'-TDE (DDD); and p,p'- and o,p'-DDE--and for m,p'-TDE, m,p'-DDE, DDMU, and DDMS, metabolites not detected at PBA but judged likely to be present (see Fig. IV-1 for structures). Throughout this report, DDT (unprefixed) refers to the technical product, sometimes designated DDTR in the literature.

Objective

The objective of this report is to provide, to those charged with assessment and amelioration of DDT contamination at Army installations, guidance on the health and environmental hazards of DDT and the ecological consequences of various actions.

II. ALTERNATIVE NAMES

DDT is the name approved by the International Standards Organization for the technical product of which p,p'-DDT is the predominant component. As used in the present report, DDT refers to the technical product or any of ten isomers or degradation products listed below.

DDT trade names: Anofex, Arkotine, Chlorophenothane, Dicophane, Estonate, Gesarol, Guesarol, Neocid, Zerdane.

p,p'-DDT: 1,1'-(2,2,2-trichloroethylidene)bis[4-chloro]benzene; α,α-bis(p-chlorophenyl)-β,β,β-trichlorethane; 2,2-bis(p-chlorophenyl)-1,1,1-trichloroethane; 4,4'-dichlorodiphenyltrichloroethane; 1,1,1-trichloro-2,2-bis(p-chlorophenyl)ethane.

o,p'-DDT: 1-chloro-2[2,2,2-trichloro-1-(4-chlorophenyl)ethyl]benzene; 1,1,1-trichloro-2-(o-chlorophenyl)-2-(p-chlorophenyl)ethane.

m,p'-DDT: 1-chloro-3[2,2,2-trichloro-1-(4-chlorophenyl)ethyl]benzene; 1,1,1-trichloro-2-(m-chlorophenyl)-2-(p-chlorophenyl)ethane.

p,p'-DDD: 1,1'-(2,2-dichloroethylidene)bis[4-chloro]benzene; 1,1-dichloro-2,2-bis(p-chlorophenyl)ethane; p,p'-TDE.

o,p'-DDD: 1-chloro-2[2,2-dichloro-1-(4-chlorophenyl)ethyl]benzene; 1,1-dichloro-2-(o-chlorophenyl)-2-(p-chlorophenyl)ethane; mitotane; o,p'-TDE.

m,p'-DDD: 1-chloro-3[2,2-dichloro-1-(4-chlorophenyl)ethyl]benzene; 1,1-dichloro-2-(m-chlorophenyl)-2-(p-chlorophenyl)ethane; m,p'-TDE.

p,p'-DDE: 1,1'-(2,2-dichloroethenylidene)bis[4-chloro]benzene; 1,1-dichloro-2,2-bis(p-chlorophenyl)ethylene.

o,p'-DDE: 1-chloro-2[2,2-dichloro-1-1(4-chlorophenyl)ethenyl]benzene; 1,1-dichloro-2-(o-chlorophenyl)-2-(p-chlorophenyl)ethylene.

DDMU: 1,1'-(2-chloroethenylidene)bis[4-chloro]benzene; 1-chloro-2,2-bis-(p-chlorophenyl)ethylene.

DDMS: 1,1'-(2-chloroethylidene)bis[4-chloro]benzene; 2-chloro-1,1-bis(p-chlorophenyl)ethane.

III. PHYSICAL AND CHEMICAL PROPERTIES[10]

p,p'-DDT:

Chemical Abstracts Service Registry Number: 50-29-3

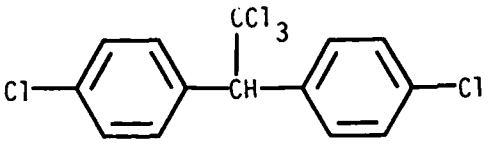
Toxic Substances List: KJ33250

Wiswesser Line Notation: GXGG YR DG&R DG

Molecular Weight: 354.48

Molecular Formula: $C_{14}H_9Cl_5$

Structural Formula:



o,p'-DDT:

Chemical Abstracts Service Registry Number: 789-02-6

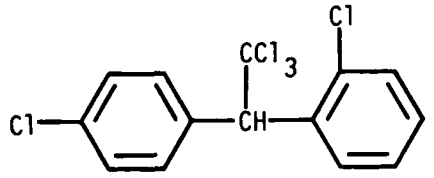
Toxic Substances List: KH7910000

Wiswesser Line Notation: GXGG YR BG&RDG

Molecular Weight: 354.48

Empirical Formula: $C_{14}H_9Cl_5$

Structural Formula:



p,p'-TDE (DDD):

Chemical Abstracts Service Registry Number: 72-54-8

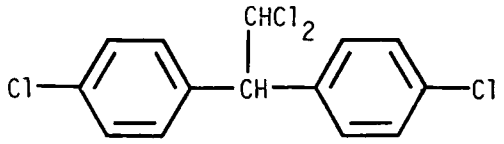
Toxic Substances List: KI0700000

Wiswesser Line Notation: GYGYR DG&R DG

Molecular Weight: 320.0

Empirical Formula: $C_{14}H_{10}Cl_4$

Structural Formula:



p,p'-DDE

Chemical Abstracts Service Registry Number: 72-55-9

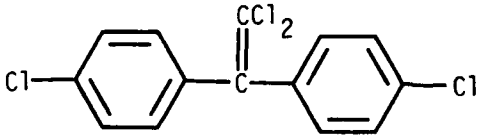
Toxic Substances List: KV9450000

Wiswesser Line Notation: GYGUYR DG&R DG

Molecular Weight: 318.0

Empirical Formula: $C_{14}H_8Cl_4$

Structural Formula:



Composition of Technical DDT

DDT is the name approved by the International Standards Organization for the technical product of which p,p'-DDT is the predominant component. Pure p,p'-DDT is a colorless crystalline solid, whereas the technical material takes the form of a white or cream-colored waxy solid or amorphous powder.

Technical DDT is a mixture of isomers containing 65 to 80% p,p'-DDT and up to 14 other components. The major impurities are o,p'-DDT (15 to 21%); p,p'-TDE (>4%; 1-(p-chlorophenyl)-2,2,2-trichloroethanol (>1.5%); traces of o,o'-DDT and m,p'-DDT; and traces of bis(p-chlorophenyl)sulfone. On exposure to sunlight or alkaline conditions, p,p'-DDT is converted to stable p,p'-DDE, which may constitute a significant fraction of any environmental sample.

Physicochemical properties of the pure substances comprising technical DDT are summarized in Table III-1.

Analysis

No attempt has been made to review analytical methods for DDT. Approved methods for detection and estimation of DDT and its derivatives in environmental samples (soil, sediment, water, and tissues) have been compiled by the U.S. Environmental Protection Agency, and are subject to frequent revision.[11,12]

IV. MAMMALIAN TOXICOLOGY

Human Exposure

DDT was introduced in 1945 for the control of malaria mosquitoes. It is a highly potent contact poison of the nervous system in insects. It is very stable, so it persists, offering continuous protection for many months after a single application. During World War II, DDT was widely used to prevent insect vector-borne disease among troops, prisoners, and refugees. DDT was applied directly to the skin and clothing in concentrations as high as 25% in powder form. Despite these massive exposures, very few, if any, authentic cases of human poisoning have been observed as a result.[13] DDT is moderately toxic to man by oral administration; Table IV-1 gives dosages and expected or observed effects in man.

Laws et al.[13,15] conducted extensive tests on 35 individuals employed in the manufacture of DDT who had been exposed from 16 to 25 years (21 years median) to amounts up to 18 mg per person per day. Physical examinations, medical histories, and liver function tests failed to reveal any evidence of an untoward effect on human health. Experimental work on human volunteers has not produced convincing evidence that DDT is harmful to man at exposure levels 100 times those likely to be encountered in the workplace or environment.[14-16]

Despite extensive studies over the past 30 years, the exact mechanism of DDT's toxic action in man is still uncertain. Based upon studies primarily

TABLE III-1. SELECTED PHYSICOCHEMICAL PROPERTIES OF COMPONENTS OF TECHNICAL DDT[10]

Property	Pure Substances Comprising Technical DDT			
	p,p'-DDT	o,p'-DDT	p,p'-TDE (DDD)	p,p'-DDE
Description	Colorless crystals	White, crystalline solid	Colorless crystals	White, crystalline solid
Melting point	108.5°C	74.2°C	109°-110°C	88.4°C
Boiling point	185°			
Solubility	Practically insoluble in water (1 µg/l), moderately soluble in hydroxylic and polar solvents, readily soluble in most aromatic and chlorinated solvents	Water, 0.085 mg/l at 25°C; soluble in fat and most organic solvents	Similar to p,p'-DDT	Water, 0.12 mg/l at 25°C; soluble in fat and most organic solvents
Molecular weight	354.5	354.5	320.0	318.0
Molecular formula	$C_{14}H_9Cl_5$	$Cl_4-H_9-Cl_5$	$C_{14}-H_{10}-Cl_4$	$C_{14}-H_8-Cl_4$
Volatility	Vapor pressure = 1.9×10^{-7} Torr at 20°C			
Chemical reactivity and stability	Dehydrochlorinated at temperatures above its melting point into ethylene derivative (DDE), a reaction catalyzed by ferric and aluminum chloride and by UV light. In solution, it is readily dehydrochlorinated by alkalis or organic bases; otherwise it is stable being unattacked by acid and alkaline permanganate and by aqueous acids and alkalis. With technical DDT, dehydrochlorination may proceed at temperatures as low as 50°C	Stable in concentrated sulfuric acid	Similar to p,p'-DDT, but it is more slowly hydrolyzed by alkalis	Stable in concentrated sulfuric acid. It may be oxidized to p,p'-dichlorobenzophenone, a reaction catalyzed by UV radiation

in laboratory animals, using relatively massive doses, it has been speculated that DDT affects the metabolism of some of the biogenic substances in the central nervous system and some of the carbohydrate-metabolizing enzymes in the uterus, kidney cortex, and liver. The microsomal enzyme systems in the liver and possibly other tissues are increased when exposure levels become sufficiently high.[17] The occurrence of enzyme induction in man at current environmental exposure levels has not been established.

Human exposure to DDT has resulted in no reported cases of cancer or other neoplasms, although carcinogenesis has been demonstrated in some laboratory animal species. Feeding DDT to men for nearly 2 years did not result in tumors,[18] and no tumors were found in men whose occupation was the manufacture, formulation, or application of DDT.[19] (However, the latency period for appearance of cancer in humans may exceed the 35 years since DDT was introduced.) The U.S. Environmental Protection Agency[20] has estimated an upper-limit lifetime cancer risk of 1 in 10^5 for males consuming 7.3×10^{-4} mg/day (10 ng/kg/day) of DDT. This estimate is derived from the observation that Jewish males in Israel have higher fat levels of DDT than males in New York State (16.33 versus 9.04 ppm) and that the lifetime incidence of nervous system cancer is correspondingly higher (1.1 versus 0.5%). It is based on the assumption that cancer resulting from DDT ingestion will be expressed in humans solely in the nervous system and on the admittedly unsupported corollary that the excess incidence of nervous system cancer results solely from excess DDT consumption.

TABLE IV-1. TOXICITY OF DDT TO MAN[a]

Dosage (mg/kg/day)	mg / 70-kg person	Remarks
Unknown[b]	--	Fatal
16-286[b]	1,100-20,000	Vomiting at higher doses, convulsions in some
6-10[b]	400-700	Moderate poisoning in some
0.5	35	Tolerated. Periods lasted 21 months with volunteers, 6.5 years with workers
0.25 (inhalation ?)	18	Tolerated by workers for 19 years

a. Adapted from Jukes.[14]
b. Precise dosage unknown.

Laboratory Animals

Acute Toxicity. Data on the acute toxicity of DDT to mammals are summarized in Table IV-2.[21] These data indicate that the short-term toxicity to mammals is moderate to high, depending on the mode of ingestion, and that DDT is generally more efficiently absorbed from the gastrointestinal tract when dissolved in an oil vehicle.

TABLE IV-2. ACUTE ORAL TOXICITY OF DDT FOR ANIMALS[a]

Species	LD_{50}, mg/kg	
	Water Suspension or Powder	Oil Solution
Rat	500-2,500	113-450
Mouse	300-1,600	100-800
Guinea pig	2,000	250-560
Rabbit	275	300-1,770
Cat		100-410
Dog		>300

a. From Hayes.[21]

Carcinogenicity. Carcinogenesis experiments have been performed in which rodents were fed DDT at concentrations ranging from 2 to 1,650 ppm.[22-31] There appear to be wide ranges in susceptibility to DDT-induced carcinogenesis for different mammalian species and strains. Other studies have found that no increase in tumors was induced by feeding DDT to golden hamsters,[32] and no tumors were induced in a small number of dogs and monkeys.[19] DDT, TDE, and DDE were tested in the National Cancer Institute Bioassay Program. The summary of their results follows.[28]

"Bioassays of technical-grade DDT, TDE, and p,p'-DDE for possible carcinogenicity were conducted using Osborne-Mendel rats and B6C3F1 mice. Each compound was administered in the feed, at either of two concentrations, to groups of 50 male and 50 female animals of each species. Twenty animals of each species and sex were placed on test as controls for the bioassay of each compound. The time-weighted average high and low dietary concentrations of DDT were, respectively, 642 and 321 ppm for male rats, 420 and 210 ppm for female rats, 44 and 22 ppm for male mice, and 175 and 87 ppm for female mice. The time-weighted average high and low dietary concentrations of TDE were, respectively, 3294 and 1647 ppm for male rats, 1700 and 850 ppm for female rats, and 822 and 411 ppm for male and female mice. The time-weighted average high and low dietary concentrations of DDE were, respectively, 839 and 437 ppm for male rats, 462 and 242 ppm for female rats, and 261 and 148 ppm for male and female mice. After the 78-week dosing period there was an additional observation period of up to 35 weeks for rats and 15 weeks for mice.

"There were significant positive associations between increased chemical concentration and accelerated mortality in female mice dosed with DDT and in both sexes of rats and in female mice dosed with DDE. This association was not demonstrated in other groups. There was, however, poor survival among control and dosed male mice used in the bioassays of DDT and DDE. In all cases adequate numbers of animals in all groups survived sufficiently long to be at risk from late-developing tumors.

"When those male rats receiving TDE and their controls were combined within each group so that the numerators of the tumor incidences represented those animals with either a follicular-cell carcinoma or a follicular-cell adenoma of the thyroid, the incidence in the low dose group was significantly higher than that in the control. There was a significant positive association between the concentration of DDE administered and the incidences of hepatocellular carcinomas in male and female mice. Among dosed rats and mice no other neoplasms occurred in statistically significant incidences when compared to their respective control groups.

"Under the conditions of these bioassays there was no evidence for the carcinogenicity of DDT in Osborne-Mendel rats or B6C3F1 mice, of TDE in female Osborne-Mendel rats or B6C3F1 mice of either sex, or of p,p'-DDE in Osborne-Mendel rats, although p,p'-DDE was hepatotoxic in Osborne-Mendel rats. The findings suggest a possible carcinogenic effect of TDE in male Osborne-Mendel rats, based on the induction of combined follicular-cell carcinomas and follicular-cell adenomas of the thyroid. Because of the variation of these tumors in control male rats in this study, the evidence does not permit a more conclusive interpretation of these lesions. p,p'-DDE was carcinogenic in B6C3F1 mice, causing hepatocellullar carcinomas in both sexes."

Mutagenicity. The fact that DDE is mutagenic in mammalian cells[33] and DDT is not suggests that the proximate carcinogen is DDE, a metabolite of DDT. It has been shown that chlorinated hydrocarbon carcinogens, such as carbon tetrachloride and dieldrin, are negative in the standard Ames test. These materials presumably require metabolic activation, possibly dehalogenation, for mutagenic activity. Because the Ames test includes only metabolic activation mediated by the liver microsomal system and dehalogenation is not so mediated, it is reasonable that pure DDT is negative in the Ames test.

Metabolism. The principal pathways for DDT metabolism are depicted in Fig. IV-1, with lesser pathways presented in Fig. IV-2. It is important to note that DDD and DDE arise by independent mechanisms and that DDE is relatively inert. Hence, environmental DDT samples will show increasing percentages of DDE with time where use of DDT has been discontinued. Equivalent metabolites arising from the o,p'-DDT isomer in technical DDT also appear in residues. The biological transformation of DDT is further discussed in the following section of this report.

V. ENVIRONMENTAL CONSIDERATIONS

The literature on the toxicology, ecology, environmental fate, and bioaccumulation of DDT is extensive and has been comprehensively reviewed elsewhere, notably by Brown,[34] Edwards,[35] Matsumura,[36] Tahori,[37] White-Stevens,[38] and Wurster and coworkers.[39-42] Information on the environmental fate of DDT and the bioaccumulation of DDT in the food chain is summarized in the following subsections.

Behavior in Soil, Water, and Air

To summarize, factors affecting the behavior of DDT in soil, water, and air include low water solubility, ease of adsorption on soil, chemical reactivity (p,p'-DDT conversion to p,p'-DDE), low vapor pressure, and ease of uptake by plants and animals. When present in soil, DDT tends to remain for years, acting as a long-lived reservoir for gradual release to surface waters and biota. When present in surface waters, DDT is assimilated rapidly by aquatic organisms and is accumulated in the food chain. Evaporation into the atmosphere also occurs. Atmospheric transport leads to low (background) concentrations over wide geographic areas. Worldwide, rainwater DDT levels fall in the range from 0.018 to 0.066 ppb.[9]

Although practically insoluble in water, DDT readily adsorbs to particulate material in aquatic systems. In addition to accumulation through the food chain, DDT may be incorporated into aquatic organisms by direct contact with DDT-containing water or through ingestion of particulate matter containing DDT.

DDT may enter an aquatic ecosystem by physical, chemical, or biological transport. Atmospheric transport and erosion of contaminated solids appear to be the most frequent routes. Eventually, the DDT tends to reach the

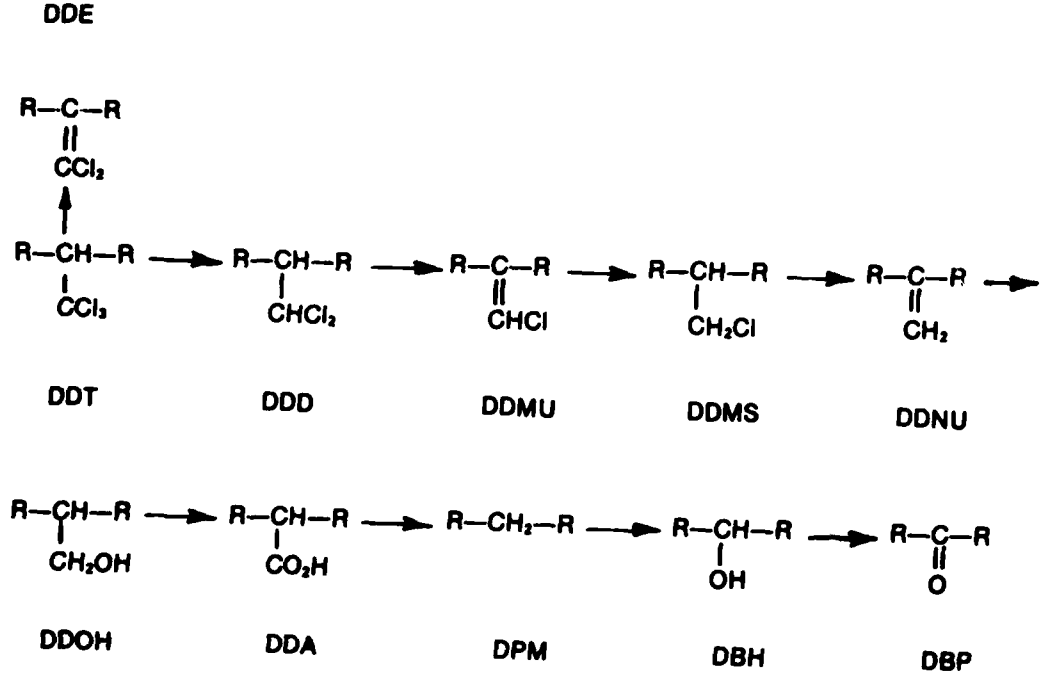
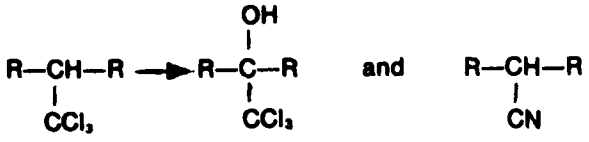
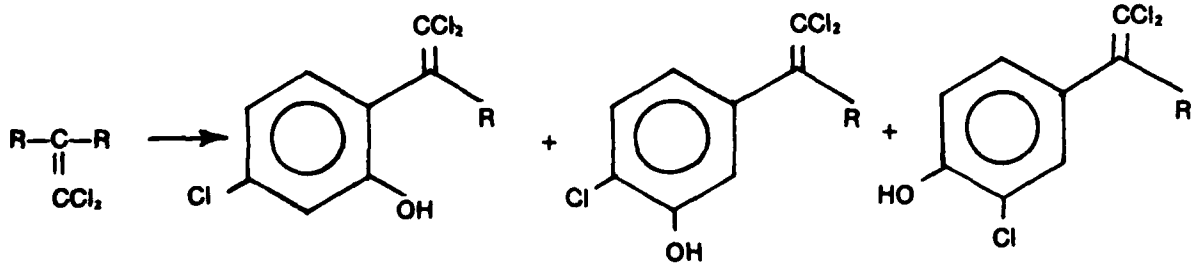


Fig. IV-1. Biodegradation of DDT (R=C_6H_4Cl)



DDT Kelthane DDCN



DDE

Fig. IV-2. Further Degradation Products of DDT and DDE (R=C_6H_4Cl)

water surface where it can co-distill with water and reenter the atmospheric cycle. As noted earlier, DDT is converted to DDE by sunlight. Various organisms also convert p,p'-DDT to p,p'-TDE and p,p'-DDE, the latter being the most abundant DDT compound in the environment. For the purpose of this review, the three compounds are considered collectively, unless specified otherwise.

The amount of DDT that runs off into water bodies depends on the degree of slope of the ground, the fineness of the soil, and the degree of vegetation cover.[43] Water transport of DDT depends on erosion runoff because DDT is strongly adsorbed to soil particles. DDT becomes so tightly bound to soil particles that it does not readily leach into groundwater.[44] Nonpolar compounds such as DDT either reach the aquatic sink adsorbed onto soil particles in the runoff or, when directly applied to water, become adsorbed onto the suspended matter.

When a pond was treated with DDT at 0.02 ppm, an effective concentration for mosquito control, the DDT disappeared from the water after 3 weeks and was found in the mud for 8 weeks after the treatment.[45] Greater amounts of DDT reach the bottom of a water body when the sedimenting material is composed of fine particles.[45]

The stability of DDT in soil has been studied by Guenzi and Beard, who have also reviewed the subject.[46-49] The rates and products of degradation are dependent on temperature, oxidation-reduction potential, and moisture content of the soil. In aerobic soils, DDT is converted to DDE by a predominantly chemical process.[48] In anaerobic soils, the products are TDE and its transformation products.[46,49] In dry aerobic soils, DDT is stable; loss is very slow by either degradation or volatilization.[46,47]

Degradation

Reviews by Fries[50] and Rhead[51] summarize much of our knowledge concerning the natural degradation of p,p'-DDT. A proposed scheme for partial biodegradation of DDT is presented in Fig. IV-1. Although the metabolites have all been identified, the pathway depicted must be considered only representative because no single organism has been found to produce all the metabolites (with the possible exception of *Aerobacter aerogenes*[52]), and it is likely that different organisms emphasize different pathways. TDE is by far the most prevalent metabolite of bacteria and fungi, whereas phytoplankton species produce small amounts of DDE only. Only TDE has been isolated from the intestinal microflora of the northern anchovy (*Engraulis mordax*).[53] Two other minor products of microbial degradation of DDT are Kelthane and DDCN (Fig. IV-2), although the latter may result in part from chemical degradation. It should be emphasized that complete biodegradation of DDT proceeding via a series of hydrodechlorination steps, as in Fig. IV-1, requires both anaerobic and aerobic conditions.

Fish that have received DDT by intravenous injection,[54] feeding,[55] or uptake from water produce TDE and DDE in various proportions in addition to some DDMU. Brook trout receiving intramuscular DDT are reported to produce only DDE.[56] DDT administered to lobsters (Homerus americanus) by intravascular or oral routes is converted to TDE, DDE, and DDA.[57] Sheridan has shown that DDT concentrated from the water is converted to TDE and DDE in the hepatopancreas of the blue crab, Callinectes sapidus.[58] Lower aquatic invertebrates convert DDT to TDE, DDE, and other metabolites, but daphnids are reported to produce only DDE.[59] Zinck and Addison have noted that p,p'-DDE is probably a metabolic dead end.[56] However, ring-hydroxylated metabolites of DDE, shown in Fig. IV-2, have been isolated from the fat of the guillemot (Uria algae) and grey seal (Halichoerus grypus).[60]

Fries has reviewed data indicating that o,p'-DDT is degraded to o,p'-TDE by mechanisms and rates similar to those for p,p'-DDT.[50] It is likely that the degradation pathways presented in Fig. IV-1 are followed.

DDT may also undergo chemical degradation. Photolysis is reported to convert DDT to TDE, DDE, DBP, and p,p'-dichlorobiphenyl, and heat also converts DDT to TDE and DDE. DDT is unchanged after 8 weeks in river water.[61]

Bioaccumulation and the Food Chain

The direct accumulation of DDT from water may, in certain cases, make the additional uptake from food insignificant. The algae and bacteria in water are very efficient concentrators of DDT; their small size, and consequently high surface-to-mass ratio, results in rapid and thorough adsorption.[34] For example, bacteria concentrated DDT from 1 ppb in water to 1,140 to 3,400 times that within 30 minutes,[62] and freshwater algae concentrated DDT from 1 ppm in water to 130 to 270 ppm in their cells within 1 week.[63] When exposed to DDT in water at concentrations between 50 and 100 ppt for 3 days, aquatic arthropods achieved increases in concentration ranging from 3,000 to 114,000 times.[64] When exposed to DDT in salt water for 2 weeks, the Atlantic croaker concentrated 0.1 ppb by 40,000 times.[65] Brown trout exposed to 2.3 ppb and given DDT-free food for 3 weeks concentrated the DDT in their tissues by 3,000 times.[66]

DDT, applied once at the rate of 1 lb/acre (1.12 kg/ha), persisted in the soil of Maine forests with little change throughout a 9-year period.[35] Robins living in the forest had higher DDT levels than those in surrounding areas, indicating a period of continuous availability of residues through the food chain, as shown in the following table:

Robin Body DDT Concentration (ppm)	Time of Analysis
13.53	1 year after treatment
4.50	3 years after treatment
3.55	9 years after treatment
0.47	Untreated areas

DDT applied to a forested area in Montana at the rate of 0.5 lb/acre (0.56 kg/ha) resulted in the following concentrations in the blue grouse.[35]

Concentration in Fat (ppm)	Time of Analysis
80	Within 1 week of spraying
22	1 year after spraying
18	2 years after spraying

Predatory or fish-eating birds usually have higher DDT residues than seed-eaters. Alaskan peregrine falcons, which feed primarily on birds, contained far higher residues than the small birds in their area.[67,68] Scaup, which feed more heavily upon animal material than mallards, accumulated residues that were 2 to 4 times as great when both were placed on a DDT-treated marsh for the same periods of time.[35]

Various small mammals were collected in Maine forests after a single application of DDT at the rate of 1 lb/acre (1.12 kg/ha).[35] In the year of treatment, shrews, mice, and voles contained an average of 15.6, 1.1, and 1.1 ppm, respectively. The relative differences between shrews and the mice and voles prevailed throughout the years after treatment. In the same areas, mink, which are carnivorous feeders like the shrews, accumulated higher total DDT residues (8.5 ppm) in the first year of treatment than hares (0.08 ppm). For areas treated seasonally with DDT, residues in small mammals increased and decreased seasonally in relation to the treatment times.

Food Chain. The bioaccumulation of DDT in the food chain is primarily a consequence of its stability and high fat solubility. In the food chain, energy is transferred from one trophic level to another. In general terms,

only about 10% of the energy in one trophic level will be transferred to the next level, and the rest will be respired or released as wastes.[69] Chemicals that are preferentially taken up by living organisms and stored for extended periods, such as DDT and its derivatives, tend to be concentrated in the food chain. Examples of DDT bioaccumulation in the food chain[69-71] are displayed graphically in Fig. V-1.

A review of the extensive literature on aquatic and terrestrial food chains is given by Brown.[34] These studies are based on measurements of the DDT content in the environment (e.g., soil and water) as well as measurements of the DDT content in tissues of various wildlife species. It would be advantageous and would simplify an environmental assessment if it were possible to relate the concentrations of DDT in the environment (viz., in soil and water) to the toxicological impact on wildlife by using established factors for DDT bioaccumulation and translocation through the food chain. Once the bioaccumulation factors were determined, it would be possible to relate toxicological effects at dietary concentrations to soil and water concentrations. This relation could be represented by bioaccumulation pathway models, such as those shown in Fig. V-2. The bioaccumulation factors given in Fig. V-2 were estimated from limited actual data for the purpose of demonstration and should be considered hypothetical.

Although attempts have been made to predict mathematically the behavior of DDT introduced into the environment,[72] the predictive capacity and utility of these models suffer from the enormous complexity of the environment. Due to the many concomitant variables (e.g., environmental site differences, species and strain differences, wide ranges in DDT base concentrations, and different lipid/water partition coefficients and equilibrium factors), it is not possible to establish categorically DDT bioaccumulation factors that have a reasonable level of significance for all ecosystems of the world. It is important to consider each environmental setting individually.

Effects on Terrestrial Animals

Mammals. No information was retrieved concerning the effects of DDT on mammalian wildlife. As noted in Section IV, acute toxicity for mammals is low in terms of likely environmental concentrations. Data from laboratory studies of mice indicate that teratogenesis and carcinogenesis could result in mammalian wildlife exposed to DDT, but this has not been confirmed by field studies. Likewise, there is no field evidence to indicate DDT-associated reproductive failure in mammals.

The high fat solubility of DDT may pose a threat to hibernating insectivores and other mammals that are exposed to high levels of dietary DDT and that release large amounts of DDT to the bloodstream from body fat during periods of high activity and scant food supply. Such DDT releases have been observed for bats containing certain chlorinated hydrocarbon insecticides in their tissues and might also occur for mammalian carnivores.

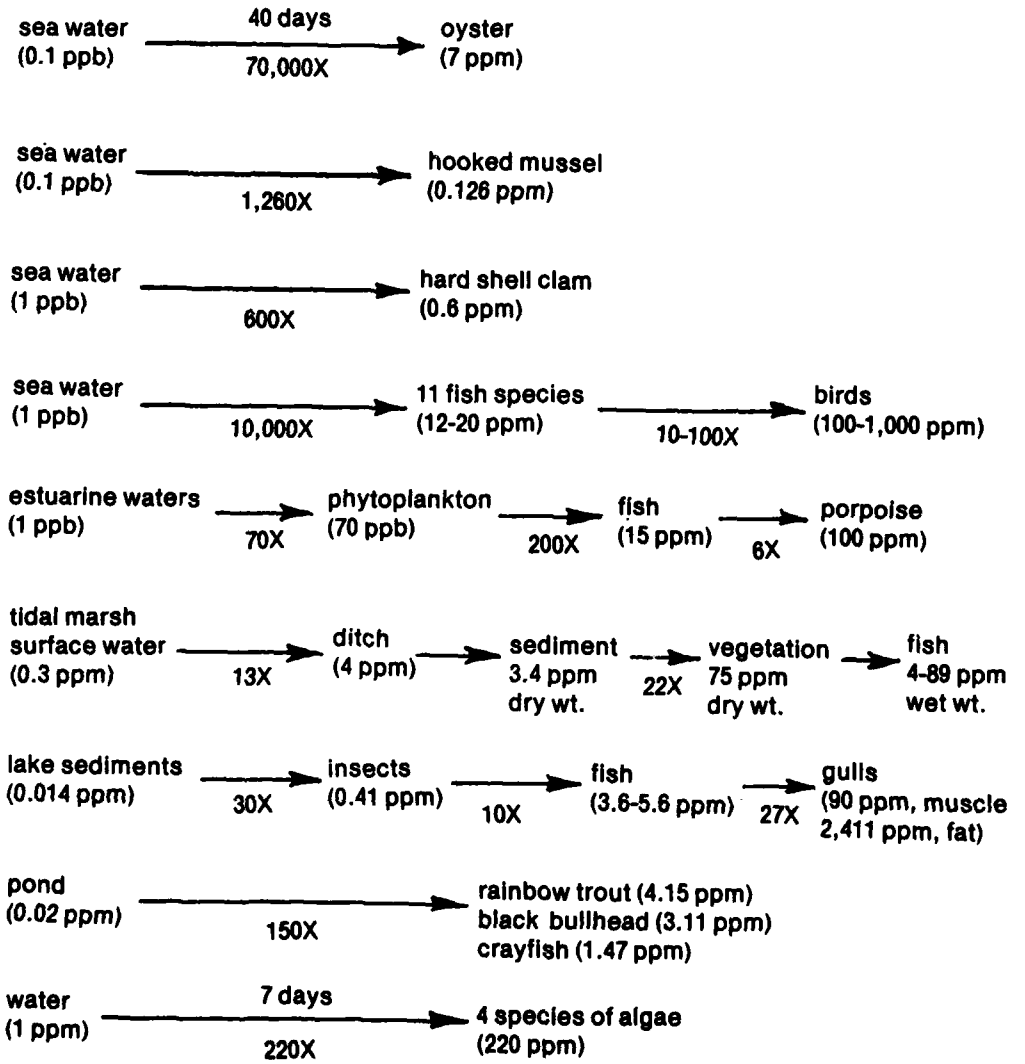
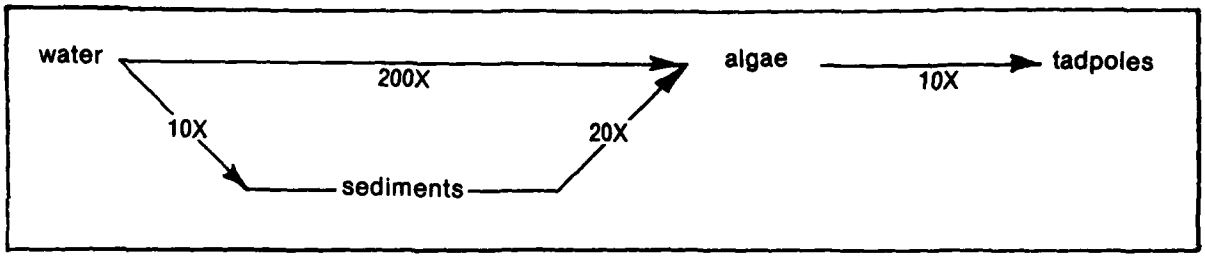
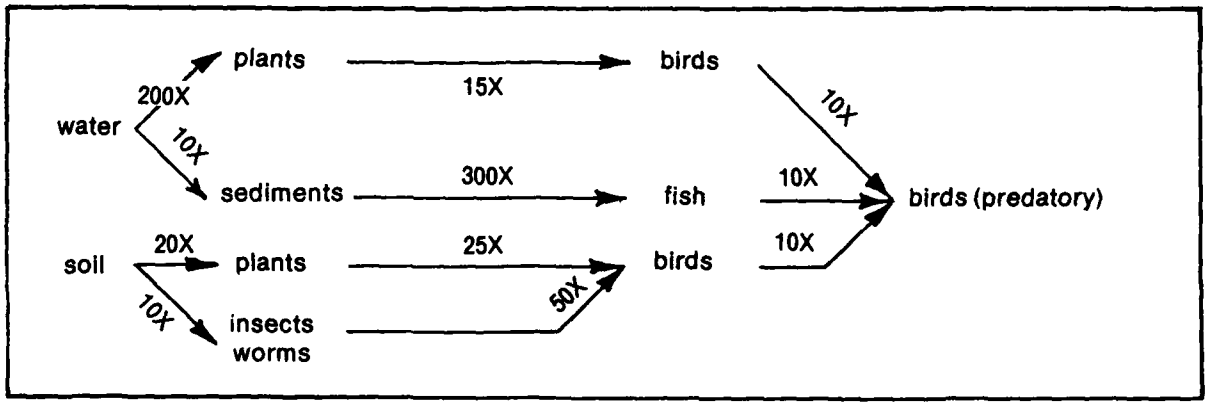


Fig. V-1. Examples of DDT Bioaccumulation

Amphibians (tadpoles)



Fish and Birds



Land-Dwelling Mammals (herbivores and carnivores)

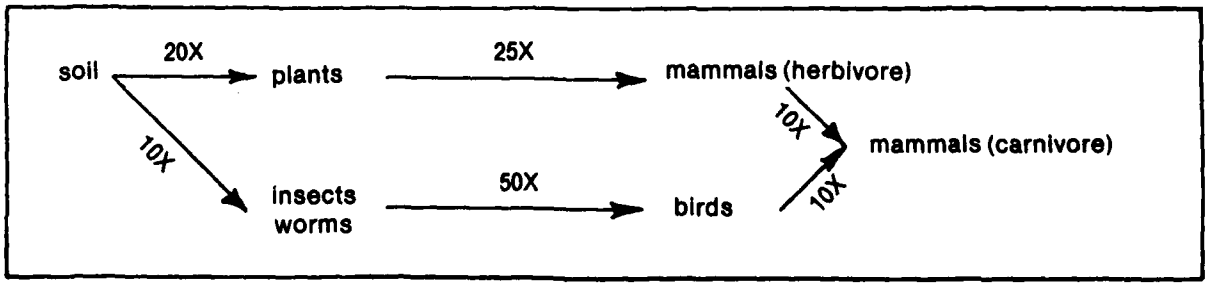


Fig. V-2. Models of Bioaccumulation Pathways

Birds. DDT and its metabolites are universally distributed so that exposure is essentially continuous, and few, if any, birds are free from these compounds. Although the acute toxicity of DDT to birds is low, direct toxic effects occur due to bioaccumulation of DDT in birds and in their food. The most serious hazard of DDT to birds is that of decreasing their reproductive capacity through eggshell thinning. It is estimated that as little as 67 ppb of DDE (the proximate agent) in the diet can cause a substantial increase in embryo mortality due to eggshell failure. The many instances of bird kills in woodlands sprayed with DDT are believed to be due to secondary poisoning by the oral route and not to contact poisoning.[73-80] However, the direct lethal toxicities of DDT to birds are low, as indicated in Table V-1.

TABLE V-1. ACUTE TOXICITY OF DDT TO BIRDS[71]

Species	Dosage Route	Toxic Effects
Mallard	Oral, capsule	LD_{50} > 2,240 mg/kg
Pheasants	Oral, capsule	LD_{50} = 1,296 mg/kg
Coturnix	Oral, capsule	LD_{50} = 841 mg/kg
Sandhill cranes	Oral, capsule	LD_{50} > 1,200 mg/kg
Mallard	Oral, 5 days	LC_{50} = 850-1,200 ppm
Pheasants	Oral, 5 days	LC_{50} = 300-700 ppm
Bobwhites	Oral, 5 days	LC_{50} = 600-1,000 ppm
Coturnix	Oral, 5 days	LC_{50} = 400-600 ppm
Pheasants	p,p'-DDT, oral	LC_{50} = 550 ppm
	Technical DDT, oral	LC_{50} = 935 ppm

The direct toxic effects of DDT to birds accompany bioaccumulation in the birds' food. Although bioaccumulation is most pronounced for predatory birds, it also can be significant for birds lower on the food chain. For example, soil contaminated with 5 to 10 ppm DDT is sufficient for earthworms to pick up 50 to 200 ppm, which could result in a lethal dose for a robin (ca. 3 mg).[34] High residues of DDT in bird fat and other tissues can be mobilized to become lethal if the birds are starved or hyperactive.[34] These processes reduce the adipose fat and release DDT into the body circulation to concentrate in the nervous system. House sparrows with DDT residues of 800 ppm in body fat displayed no adverse physiological signs if well fed, but died if not well fed; the DDT mobilization engendered tremors that further reduced fat and sent lethal concentrations into nerve and brain.[81,82] The minimum content of DDT in the brain at which death occurs is 50 ppm for American robins and 60 ppm for house sparrows,[81] while it is 14 ppm for female ring-necked pheasants.[83]

Concerning reproductive effects, a 30% decline in breeding pairs of the fish-eating osprey on the coast of Connecticut in 1963 was found to be associated with a high body content of DDT residues, especially DDE.[84,85] The reproductive failure was later related to a reduction of the eggshell thickness due to contamination of the eggs by DDT and its metabolites. Feeding experiments with mallards showed that 40 ppm of DDE in the diet resulted in frequent shell cracking, leading to 40% embryo mortality and 75% reduction in duckling production.[85] A concentration of 20 ppm of DDT in the diet of mallards resulted in 20% reduction in eggshell thickness.[86] A concentration of 10 ppm of DDE in the diet caused 25% shell thinning in the American sparrow hawk,[87] 13% in the screech owl,[88] and 18 to 29% in the black duck.[89] DDT in the diet of pheasants had little or no effect on egg production or fertility, but hatchability and chick survival were reduced at concentrations of 100 ppm or more.[90] In bobwhite quail on a diet containing 100 ppm, egg production was normal, but fertility and hatchability were reduced, and chick survival was eventually zero.[91] In addition, high dietary doses of DDT have reduced sperm production in cockerels[92] and the bald eagle.[93]

It is generally accepted that DDE is the major shell-thinning factor, because a linear inverse relationship between shell thickness and DDE content of the egg has been demonstrated for the prairie falcon, herring gull, double-crested cormorant, brown pelican, and peregrine falcon.[94,95] In general, whenever the residues induced eggshell thinning more than 10% below the normal thickness, that bird population would decline.[96] Concentrations of DDE that elicit this effect in various species of birds are listed in Table V-2. The bird prey for one population of peregrine falcons have whole body residues of 0.3 to 6.0 ppm DDE, whereas the fat and eggs of the falcons contain 560 and 15 ppm DDE, respectively.[67] This concentration factor of 2.5 to 50 for eggs, combined with an observed concentration of 8 ppm in peregrine falcon eggs for onset of reproductive failure (Table V-2), corresponds to a dietary limit of 0.16 to 3.2 ppm. If the same concentration factor is arbitrarily assumed for other birds, then the dietary threshold for reproductive failure would fall in the range of 1.6 to 32 ppm for the great blue heron, 0.05 to 1.0 for the osprey, and 0.02 to 0.4 for the brown pelican. Based on the latter two birds being fish-eaters, it appears that substantially lower levels of DDE (and hence DDT) in fish may be required to assure the survival of these birds than to protect human health.

Effects on Aquatic Organisms

Because the proportions of the various isomers and metabolites of DDT in different environmental samples are quite distinct, and because the toxicological data base for aquatic organisms is large, every effort has been made to identify the toxic effects associated with each specific isomer or metabolite throughout this section.

TABLE V-2. CONCENTRATIONS OF DDE IN BIRD EGGS RESULTING IN 10% REDUCTION IN NORMAL SHELL THICKNESS

Bird Species	DDE Concentration in Eggs (ppm wet weight)	Reference
Double-crested cormorant	20	95
Prairie falcon	7	96
Brown pelican	1	97
Great blue heron	80	71
Herring gull	70	71
Atlantic gannet	25	71
White pelican	10	71
Fish-eating osprey	2.4	99
Alaskan peregrine falcon	8	68

Fish. The acute toxicity of p,p'-DDT to fishes has been reviewed by Pimentel[71] and others.[100-103] Some representative data are presented in Table V-3, which shows that the 96-hr LC_{50} for most fishes falls between 1 and 20 µg/l. Fish and Wildlife Service investigators at the Fish-Pesticide Research Laboratory in Columbia, Missouri, report 96-hr LC_{50}'s in this range for 18 common freshwater fishes.[110] They also report that p,p'-DDT is roughly three times as toxic to bluegills (Lepomis macrochirus) at 7°C as at 24°C. Macek notes that for most common formulations containing DDT and other pesticides, acute toxicities to bluegills are additive.[115] The low LC_{50} values may be due to the rapid uptake and concentration of DDT in fish. For example, brown trout exposed to 2 ppb DDT can concentrate it about 500 times in the gill tissues and about 3,000 times in the muscle.[66] The gills of 2-lb brown trout pass about 700 liters of water per day.[116] In addition, certain fish, such as catfish, appear to be fairly tolerant to DDT under laboratory conditions, whereas in a natural setting they may succumb through bottom-feeding at the sediment level.

Sublethal concentrations of DDT to adult fish may lower their reproductive success because DDT accumulates in egg yolk and kills the fry shortly after they hatch from contaminated eggs.[56,102] The DDT is passed into the egg yolk, the embryo develops and hatches, and at the stage of

TABLE V-3. ACUTE TOXICITY OF p,p'-DDT TO FISHES BY STATIC BIOASSAY

Species	Temp. (°C)	Exposure Time (hr)	LC_{50} (µg/l)	Reference
Rainbow trout				
Salmo gairdneri	13	96	7 (5-10)[a]	104
	16	96	3.8 (3.4-4.3)	105
	12.9	96	1.72 (1.42-2.09)	106
		96	28	107
		360	0.26	108
Brown trout				
Salmo trutta	13	96	2 (1-3)	104
Brook trout				
Salvelinus fontinalis	13	96	7.4-11.9	106
Cutthroat trout				
Salmo clarki	13	96	0.85-1.37	106
Coho salmon				
Oncorhynchus kisutch	13	96	11.3-18.5	106
	9-11	96	13	109
	13	96	4 (3-6)	104
Chinook salmon				
Oncorhynchus tshawytscha[b,c]	13	96	0.68	110
Bluegill				
Lepomis macrochirus	18	96	8 (6-10)	104
	24	96	2.2 (1.8-2.6)	105
	23	96	7	111
Redear sunfish				
Lepomis microlophus	18	96	5 (3.9)	104
Largemouth bass				
Micropterus salmoides	18	96	2 (1-3)	104
Goldfish				
Carassius auratus	18	96	21 (14-30)	104
	24	96	9.8 (7.3-13.2)	105
Carassius carassius		96	25	107

TABLE V-3. (Cont.)

Species	Temp. (°C)	Exposure Time (hr)	LC_{50} (µg/l)	Reference
Carp				
Cyprinus carpio	18	96	10 (7-13)	104
Fathead minnow				
Pimephales promelas	18	96	19 (13-27)	104
Channel catfish				
Ictalurus punctatus	18	96	16 (9-28)	104
	24	96	13.5 (9-20)	105
	26	24	34	111
Black bullhead				
Ictalurus melas	18	96	5 (3-7)	104
Yellow perch				
Perca flavescens	18	96	9 (7-11)	104
Mosquitofish				
Gambusia affinis		96	20	112
		96	27	111
Guppy				
Poecilia reticulata		96	3	112
Mozambique mouthbreeder				
Tilapia mossambica		96	7	112
Aholehole				
Kuhlia sandvicensis[b]		96	3.9	112
Nehu				
Stolephorus purpureus[b]		12	1.0	112
Striped bass				
Roccus (Morone) saxatilis[d]	17	96	0.53 (0.38-0.84)	113
Roccus (Morone) saxatilis[b]	13	96	0.9	110

TABLE V-3. (Cont.)

Species	Temp. (°C)	Exposure Time (hr)	LC_{50} (µg/l)	Reference
Shiner perch				
Cymatogaster aggregata	13	96	7.6	114
	17	96	0.45	110
Dwarf perch				
Micrometrus minimus[d]	13	96	4.6	114
Micrometrus minimus[b,c]	18	96	0.26	110
White seaperch				
Phanerodon furcatus[b,c]	19	96	0.74	110
English sole				
Parophrys vetulus[b,c]	16	96	0.91	110
Pacific staghorn sculpin				
Leptocottus armatus[b,c]	19	96	0.98	110
Rubberlip seaperch				
Rhacochilus toxotes[b,c]	19	96	1.01	110
Goby				
Acanthrogobius flavimanus[b,c]	19	96	2.40	110
Speckled sanddab				
Citharichthys stigmaeus[b,c]	19	24	10.0	110
	19	48	7.2	110
	19	96	3.7	110
	19	120	1.7	110
	19	144	0.9	110

a. Numbers in parentheses are 95% confidence interval.
b. Seawater.
c. Dynamic bioassay.
d. Brackish water.

final yolk sac adsorption after hatching, the fry will die if the DDT concentration in the yolk is sufficiently high.[117,118] This phenomenon was first observed in the lake trout of Lake George, New York;[119] and later at Jasper, Alberta;[120] Lake Taupo, New Zealand;[121] Lake Michigan;[122] Sebago Lake, Maine;[123] and other locations.[34] Data for studies in these areas are listed in Table V-4 and indicate that DDT concentrations in water as low as 0.004 ppb can cause a significant increase in sac-fry mortality.

No reports were recovered describing systematic studies of the chronic effects of DDT on life stages of fishes. A DDT concentration of 5 mg/l has been shown to result in 48% mortality of carp embryos reared in vitro.[125] Exposure of Atlantic salmon (Salmo salar) eggs to 50 µg/l of DDT at gastrulation retards behavioral development in the newly hatched alevins.[126] The coughing frequency in juvenile coho salmon was found to be enhanced significantly after 4 days' exposure at a sublethal concentration of 5 µg/l.[109] High sublethal (0.3 to 3 µg/l) levels of DDT have been found to result in loss of glycogen and other pathological changes in the liver of zebrafish (Brachydanio rerio) and, to a much lesser extent, of guppy.[127] Interrupted exposure of salmonid fishes to high sublethal concentrations of DDT is reported to raise the lower lethal temperatures, alter the temperature selectivity, diminish learning ability, and affect the central nervous system in general.[128-130] Continuous exposure to 10 µg/l for 4 days is said to alter the exploratory[131] and locomotor[132] behavior of goldfish (Carassius auratus).

Desaiah et al. have presented evidence for 50% or greater inhibition of activity of mitochondrial Mg^{2+} ATPase, an important energy-linked enzyme, in brain homogenates of fathead minnows chronically exposed to DDT at a level of 0.5 µg/l for 266 days.[133] There is also a substantial, although lesser, drop in gill Na-K-ATPase activity. The latter enzyme functions in osmoregulation in marine fishes, and in this regard, Leadem et al. have found that seawater-acclimated rainbow trout receiving 2.75 mg/kg DDT/48 hr in their diet exhibit impaired osmoregulation as well as inhibition of gill Na-K-ATPase activity.[134] Kinter et al. have reported similar disruption of osmoregulation in two marine species, mummichog (Fundulus heteroclitus) and American eel (Anguilla rostrata), at lethal DDT concentrations.[135] Weisbart and Feiner report that goldfish (C. auratus) exposed to DDT at a level of 17.5 to 35 µg/l exhibited no clear evidence for impaired osmoregulation.[136] This agrees with the observation of Leadem et al. that osmoregulation is unimpaired by DDT in the diet of the freshwater rainbow trout.

The 90-dose (30-day) oral LD_{50} for juvenile coho and chinook (Oncorhynchus tshawytscha) salmon have been reported as 64 and 27.5 mg/kg/day, respectively.[137] Sublethal oral doses may result in loss of light discrimination in rainbow trout.[138]

TABLE V-4. SAC-FRY MORTALITY FOR VARIOUS FISH SPECIES

Fish Species	DDT Conc. in Eggs (ppm)	Effect	Estimated DDT Conc. in Water[a] (ppb)	Reference
Lake trout	3-355	Fry containing more than 3 ppm died at the time of final adsorption of the yolk sac	0.03	119
Brook, rainbow, and cutthroat trout	>0.4	30 to 90% sac-fry mortality	>0.004	120
Rainbow trout	5	45% sac-fry mortality	0.05	121
Coho salmon	1.1-2.8	15 to 75% sac-fry mortality, respectively	0.011-0.028	122

a. The DDT concentrations in water were estimated using a concentration factor of 100,000. The factor was based on data from a study with fathead minnows reared in 2 ppb DDT for a 9-month period. DDT concentrated in their eggs to more than 100,000 times the water concentration.[124] This is the only long-term study giving both egg and water concentrations that could be found in the literature.

Fragmentary evidence indicates that o,p'-DDT is less toxic to fish than p,p'-DDT. The 96-hr LC_{50} for goldfish (C. auratus), as measured by Ginsburg,[139] is 1.0 mg/l for o,p'-DDT, compared with about 0.06 mg/l for the p,p'-isomer. Gardner reports that brook trout fingerlings are unharmed by 24-hr exposure to o,p'-DDT at a concentration of 0.05 mg/l, although there is a noticeable effect on temperature selection at 0.02 mg/l, i.e., cooler water is preferred by exposed fish.[128] According to Alabaster, the 24-hr LC_{50} for harlequin fish (Rasbora heteromorpha) is 30 µg/l for o,p'-DDT, compared with 13 µg/l for the p,p'-isomer.[140] No information was retrieved for m,p'-DDT.

Toxicity data for p,p'-TDE (DDD) have been reviewed by McKee and Wolfe.[100] Fragmentary evidence, presented in Table V-5, indicates that TDE is highly toxic to fishes, although perhaps a half order of magnitude less toxic than p,p'-DDT. Gardner has demonstrated that high sublethal levels of TDE affect temperature selection by fingerling brook trout.[128] He further reports that brook trout are unharmed by 24-hr exposure to o,p'-TDE at a concentration of 50 µg/l, although there is some effect on temperature selection at 10 µg/l.[128] No information was retrieved for m,p'-TDE.

Gardner has found that brook trout are unharmed by exposure to 50 µg/l of p,p'-DDE for 24 hours and that there is almost no effect on temperature selection.[128] Applegate et al. report that rainbow trout, bluegills, and the larvae of sea lampreys (Petromyzon marinus) are unaffected by 24-hr exposure to DDE at 5 mg/l and 55°F.[141] Others report 96-hr LC_{50}'s of 10 to 100 µg/l for bluegills and rainbow trout at 24° and 13°C, respectively.[110] No information was retrieved for m,p'-DDE.

Reptiles. No quantitative toxicity data were recovered, but Stickel has stated that the box turtle population of a Maryland forest was not noticeably affected by DDT applied at a dosage of 2 lb/acre (2.2 kg/ha).[142] Evidence both for and against loss of reptiles through land application of DDT is summarized by McKee and Wolfe.[100] Direct treatment of ponds at DDT concentrations of 2 ppm or more has killed water snakes and turtles.[143] In the Brazos River floodplain of Texas, where cottonfields had been heavily treated with DDT, the average residues in the fat bodies of aquatic snakes were DDE, 510 ppm; TDE, 1.5 ppm; and DDT, 16.0 ppm.[35] The DDT residues in the brain did not exceed 1.5 ppm, and fat-body residues in terrestrial snakes were much lower than in aquatic snakes.[144]

In vitro treatment of cellular fractions from various tissues of six species of terrestrial turtles resulted in negligible to substantial inhibition of Mg^{2+}-, (Na^+, K^+)-, and (Na^+, K^+, Mg^{2+})-dependent ATPase at DDT levels of 2 to 76 mg/l.[145,146] Similarly, in vitro treatment of cellular fractions from various tissues of the red-eared turtle, Chrysemys scripta elegans, resulted in negligible to substantial inhibition of ATPase at TDE or DDE levels of 2 to 76 mg/l.[146]

Amphibians. For tadpoles of Fowler's toad (Bufo woodhousii fowleri) and the chorus frog (Pseudacris triseriata), Sanders reports 24-hr LC_{50} habitat water values of 2.4 and 1.4 mg/l, respectively,[147] whereas a 96-hr LC_{50} of 0.27 mg/l for bullfrog tadpoles is reported by Carter and Graves.[111] Another reference gives a 96-hr LC_{50} of 0.8 mg/l for 5-week-old tadpoles of P. triseriata and 0.74, 1.0, 0.1, and 0.038 mg/l for B. woodhousii tadpoles of 1, 4 to 5, 6, and 7 weeks, respectively.[110] These data are summarized in Table V-6. A lethal concentration of 0.15 mg/l is given for Bufo bufo tadpoles.[148] Some relative and highly ambiguous toxicity assessments based on DDT application data have been provided by Pimentel[71] and Cooke.[149]

TABLE V-5. ACUTE TOXICITY OF p,p'-TDE (DDD) TO FISHES

Species	Temp. (°C)	Exposure Time (hr)	LC_{50} (µg/l)	Reference
Goldfish[a]			1,000	100
Channel catfish[a]	20	96	<2,600	100
	18	96	15,000	110
Bluegill[a]	24	96	30[b]	100
			>10	110
Striped bass[c] *Morone saxatilis*	17	96	2.5 (1.6-4)[d]	113
Brook trout *Salvelinus fontinalis*	10	24	45	128
Rainbow trout[a]	13	96	43-93	110
Fathead minnow[a]	18	96	1,000-10,000	110
Largemouth bass[a]	18	96	39	110
Walleye[a]	18	96	10-100	110

a. Species not given.
b. Toxicity threshold.
c. Bioassay in saline water.
d. Numbers in parentheses are 95% confidence interval.

Field studies showed that 0.1 kg DDT/ha applied as an emulsion did not kill tadpoles, but 1.0 kg DDT/ha achieved 80% mortality in two days.[169] The toxic effects on frog and toad tadpoles of DDT sprayed in the field at 0.4 to 0.5 kg/ha is given by Cooke.[169] The DDT was sprayed on the water surface, as would be appropriate to kill mosquito larvae. Five water sites were monitored. DDT concentrations in surface water and in water at a depth of 20 cm decreased as the size of the water body increased, to the extent that DDT was not detected (<0.02 ppb) in water from the two larger sites. The DDT residue concentrations and the behavioral and morphological abnormalities of the tadpoles for the three smaller sites are summarized in Table V-7. The residues were measured one day after spraying. It is important to note that virtually all of the DDT sprayed was taken up by algae or incorporated elsewhere within only 3 days. Hence, the increases in

the DDT levels in the tadpoles after the third day may be due to direct ingestion of DDT-contaminated algae. A schematic diagram of behavioral abnormalities versus time after spraying DDT is given in Fig. V-3. Average DDT concentrations are derived from data of Table V-7 assuming that there is a linear gradient of concentration with depth.

TABLE V-6. TOXICITY OF DDT TO TADPOLES

Species	Exposure Time (hr)	LC_{50} (mg/l)	Reference
Bullfrog	96	0.27	111
Chorus frog	24	1.4	147
	96	0.8	110
Fowler's toad	24	2.4	147
(1 week)	96	0.74	110
(4-5 weeks)	96	1.0	110
(6 weeks)	96	0.1	110
(7 weeks)	96	0.038	110

For tadpoles of Pseudacris triseriata and Bufo exposed to p,p'-TDE, 96-hr LC_{50}'s of 100 to 1,000 and 18 µg/l, respectively, have been reported.[110] No other information concerning isomers or metabolites was retrieved.

Invertebrates. For the most part, only references dealing with nontarget species were retrieved. Toxicity data for arthropods, taken from Pimentel's review,[71] Malina's review,[150] and some recent papers, are summarized in Table V-8, which shows that marine and freshwater species demonstrate about the same order of acute sensitivity to DDT as fishes, although ostracods appear to be more resistant. There is also evidence for impaired reproductive capability in ostracods,[153] brine shrimp,[154] and Daphnia at sublethal levels.[152] Ingested DDT has been shown to be harmful to crayfish (Procambarus clarkii), blue crabs (Callinectes sapidus),[157,158] and fiddler crabs (Uca pugnax),[159] but the reported data are not readily quantified. Larvae of two caddisflies (Hydropsyche pellucidula and H. instabilis) have been found to construct irregular webs when exposed to DDT at sublethal levels (2.5 µg/l).[160] In field studies, it was found that when an unprotected stream was sprayed directly with 1 lb/acre (1.1 kg/ha), nymphs of all species of mayflies were exterminated and larvae of every species of caddisfly were affected to some extent.[84]

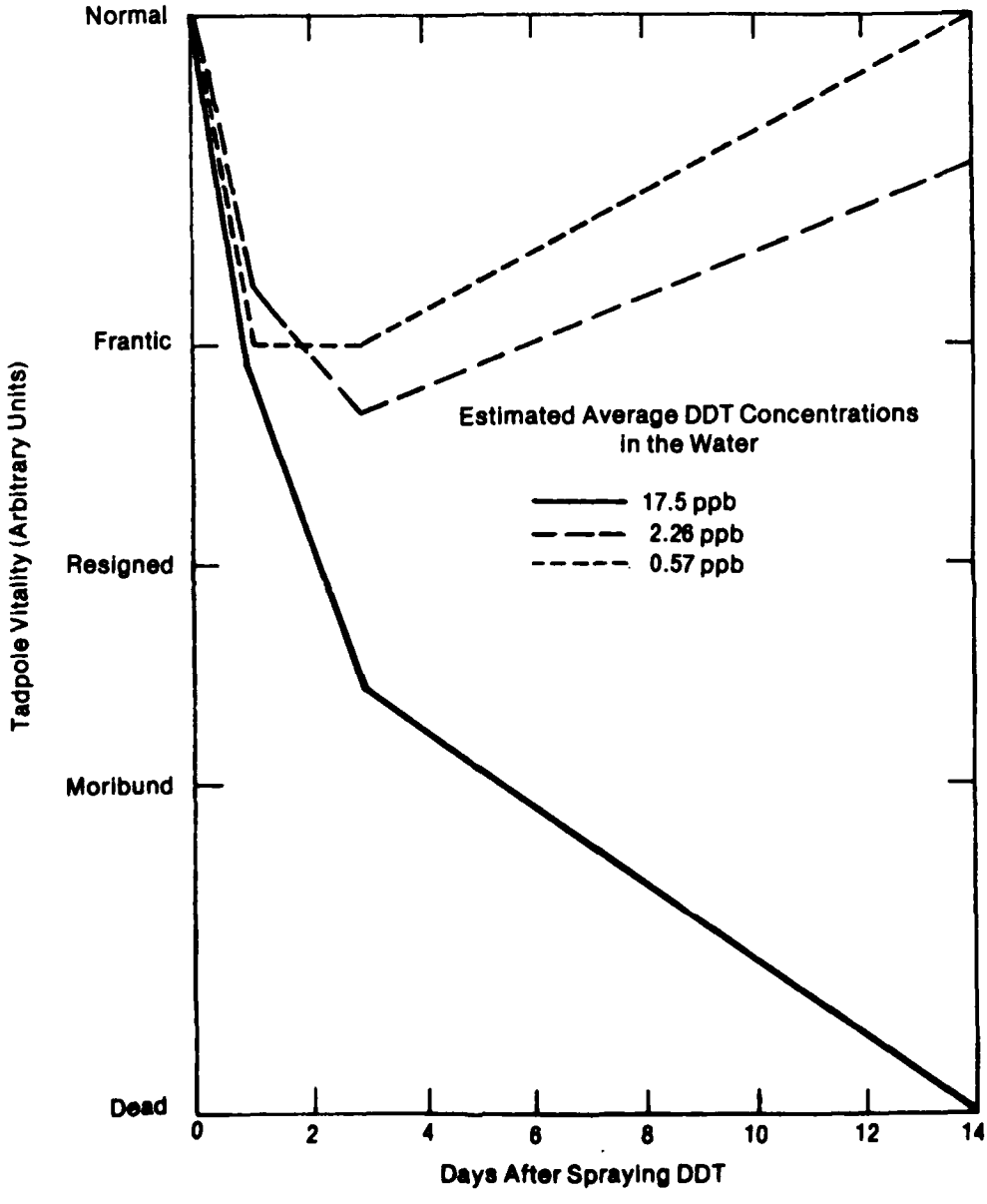


Fig. V-3. Effects of DDT on Frog Tadpoles

TABLE V-7. BEHAVIOR AND MORPHOLOGICAL ABNORMALITIES OF TADPOLES EXPOSED TO DDT

Site	Total Water Residues One Day After Spraying[a] (µg/l)		DDT Levels in Tadpoles (µg/g)			Behavioral and Morphological Abnormalities					
						Day 1		Day 3		Day 14	
	Surface	20 cm below Surface	Day 1	Day 2	Day 14[b]	Behavior	Abnormalities	Behavioral	Abnormalities	Behavior	Abnormalities
Small Ditch	36.2	17.5	7.90	6.52	None left	Frantic[c]	None	Resigned	All survivors-abnormal snouts, 4/8 dead tadpoles had tails laterally curled to *left*	No survivors	--
			1.21	3.27	None left	Frantic	None	Some frantic, some resigned	All survivors-abnormal snouts	No survivors	--
Larger Ditch	10.5	4.9	0.70	0.77	0.24	Some normal, Some frantic	None	Few normal, most frantic, few resigned	None	4 normal, 1 moribund	1 downcurved in body and tail
			0.99	0.64	0.38	Frantic	None	Most frantic, few resigned	2 dead tadpoles with upturned tails	13 normal, 3 moribund	1 with abnormal snout, 2 downcurved
Pool	2.7	1.9	1.16	0.52	1.07	Frantic	None	Frantic	None	Normal	None
			0.83	1.23	1.97	Frantic	None	Frantic	None	Normal	None

a. Other than the 0.19 µg/l from below-surface sample of small ditch taken on Day 3, no residues were detected in water samples on Days 3 and 14, ...conc. <0.02 µg/l; spraying was uneven; two large sites had no detectable (<0.02 µg/l) DDT in water.

b. Increases probably due to eating DDT-contaminated algae.

c. Frantic = hyperactivity, greatly excited, frenzied
Resigned = passive, submissive
Moribund = dying.

Progressive stages of abnormal behavior.
(All showed slow rate of metamorphosis.)

TABLE V-8. TOXICITY OF p,p'-DDT TO ARTHROPODS

Species	Exposure Time (hr)	EC_{50} or LC_{50} (µg/l)	Reference
Sand shrimp	24	3	71
Seed shrimp			
Cypridopsis vidua	48	54	151
Glass shrimp			
Palaemonetes kadiakensis	48	4.2	151
	96	2.3	110
Grass shrimp	24	12	71
Stonefly			
Pteronarcella badia	24	12	71
	96	1.9	110
Classenia sabulosa	24	16	71
	96	10	150
	96	3.5	110
Pteronarcys californica	24	41	71
	48	19	71
	96	100	150
	96	7.0	110
Acroneuria pacifica	96	180	150
Waterflea			
Daphnia pulex	48	0.36-3.6	71
Daphnia magna	48	4	151
	96	1	150
	366	0.67	152
Simocephalus serrulatus	48	0.4	71
Ostracod			
Cyprinotus incongruens	48	1,300[a]	153
Cypridopsis vidua	48	230[a]	153
Brine shrimp			
Artemia selina	48	46[a]	154

TABLE V-8 (Cont.)

Species	Exposure Time (hr)	EC_{50} or LC_{50} (µg/l)	Reference
Crayfish			
Procambarus acutas	48	3(7.2)[c]	155
Orconectes nais	96	0.24	108
(10-week)	96	30	110
Damselfly			
Ishnura verticalis	48	22.5	151
	96	1.0	110
Sowbug			
Asellus brevicaudus	48	4.7	151
	96	4.0	110
Amphipod			
Gammarus lacustris	24	4.7	71
	48	2.1	71
	96	1.0	110
Gammarus fasciatus	48	3.6	151
	96	3.2	110
Hermit crab[a]	24	7	71
Purple shore crab			
Hemigrapsus nudus	96	1.85	110
Market crab			
Cancer magister	96	4.6	110
Brown shrimp			
Crangon crangon	48	3.3-10	156

a. Species not given.
b. Extrapolated from author's data.
c. Value in parentheses for crayfish acclimated to natural, DDT-contaminated water of an unspecified concentration.

Although mollusks are not so readily killed by DDT, the growth of eastern oysters is reported to be reduced significantly (and reversibly) at a level of 0.1 µg/l,[71] and survival of the larvae of the American oyster (Crassostrea virginica) is diminished by 20% at a level of 25 µg/l.[161] Annelids are so insensitive to DDT intoxication as to present a dietary hazard to predator organisms. The 96-hr LC_{50} for the buffalo leach (Hirudinari manillensis) exceeds 100 mg/l,[162] and tubeficid worms (Branchiura sowerbyi) are said to exhibit no mortality after 72 hours at a level of 4 mg/l and 21°C, although they are completely destroyed when exposed to the same concentration at 4.4° and 32.2°C.[163] The extrapolated 96-hr LC_{50} for a planarian (Polycelis felina) is 1.26 mg/l at 6.5°C.[164] Earlier data for invertebrates have been reviewed by McKee and Wolfe,[100] and some additional toxicity data are contained in Reference 108.

Fragmentary evidence, presented in Table V-9, indicates that o,p'- and m,p'-DDT may be less acutely toxic to mosquito larvae than the p,p'-isomer. No information concerning nontarget species was retrieved.

TABLE V-9. TOXICITY OF DDT ISOMERS TO MOSQUITO LARVAE

	Anopheles quadrimaculatus[a] [165,166]		Aedes aegypti[139]
Isomer	24-hr LC_{50} (µg/l)	48-hr LC_{50} (µg/l)	96-hr LC_{50} (µg/l)
p,p'-DDT	2.5	< 2.5	11
o,p'-DDT	15	10	350
m,p'-DDT	15	<10	

a. 4th instar.

Data relating the acute toxicity of p,p'-TDE to arthropods are summarized in Table V-10. Comparison of Tables V-8 and V-10 reveals that for many arthropods TDE is equal to or greater in toxicity than DDT. McKee and Wolfe have reviewed pesticide application data and note that the larvae of Chaoborus (phantom midge) and gnats are "controlled" at 13 to 14 µg/l and chironomid (midge) larvae are temporarily eliminated.[100] With a 96-hr LC_{50} of 740 µg/l, TDE is slightly more toxic to the freshwater planarian Polycelis felina than DDT.[164]

The 96-hr LC_{50} of p,p'-DDE to the freshwater planarian Polycelis felina is 1.23 mg/l, only slightly more than the corresponding value for DDT.[164] No further information was retrieved concerning isomers or metabolites.

TABLE V-10. ACUTE TOXICITY OF p,p'-TDE (DDD) TO ARTHROPODS

Species	Exposure Time (hr)	EC_{50} or LC_{50} (µg/l)	Reference
Amphipod			
Gammarus lacustris	96	0.64	108
Gammarus fasciatus	96	0.86	108
Sowbug			
Asellus brevicaudus	96	10	108
Waterflea			
Daphnia magna	72	0.1[a]	167
Daphnia pulex	48	3.2	108
Simocephalus serrulatus	48	4.5	108
Glass shrimp			
Palaemonetes kadiakensis	96	0.68	108
	72	0.1[a]	167
Mosquito (4th instar)			
Anopheles quadrimaculatus	24	2	168
Stonefly			
Pteronarcys californica	96	380	108

a. Sublethal effects.

Effects on Microorganisms

Luard has reviewed, in part, the literature on DDT toxicity to freshwater and marine phytoplankton, and notes evidence for a wide range of sensitivities.[169] Other data are contained in Reference 108. A few marine species exhibit inhibition of photosynthesis at 1 to 10 µg/l, but in general there is no effect on growth at levels below 100 µg/l (see also Pimentel[71]). A recent study shows an even higher level of resistance in Euglena.[170]

Bacteria also appear to be resistant to DDT. The growth of Bacillus megaterium in nutrient media is unaffected by 100 mg/l of DDT, although the death rate of resting cells is measurably enhanced at 1 mg/l.[171] Growth of Azotobacter chroococcum is said to be unchanged in the presence of 400 mg/l.[172] The growth rates of Pseudomonas fluorescens and Staphylococcus aureus, but not Escherichia coli, are noticeably inhibited at 50 mg/l.[173] It is probably safe to assume that microorganisms will be unaffected by p,p'-DDT at levels selected to protect fish and invertebrates.

The chemolithotrophic nitrofier, Nitrobacter agilis, is completely inhibited by TDE at a concentration of 10 mg/l and measurably inhibited at 0.1 mg/l.[174]

DDE (as well as DDT) at a concentration of 10^{-6} to 10^{-5} M (0.35 to 3.5 mg/l) is said to inhibit photosynthetic electron transport in the green algae Codium fragile and Chaetomorpha area and in isolated chloroplasts.[169] DDE is reported to be more toxic than DDT to the marine dinoflagellate Exuviella baltica, causing significant growth inhibition at levels as low as 0.1 µg/l.[175] No other information concerning isomers or metabolites was retrieved.

VI. STANDARDS AND CRITERIA FOR DDT

Air

Threshold limit values for the workroom environment:[176]

Time-weighted average: 1 mg/m^3
Short-term exposure limit: 3 mg/m^3

Drinking Water and Food

Allowable daily intake:[9] 0.005 mg/kg/day

Maximum concentration in fish and agricultural products for interstate commerce:[177] 5 ppm

Water for Aquatic Life

EPA recommended criterion:[20] 0.00023 µg/l (24-hr average) and 0.00041 (not to be exceeded at any time)

VII. EFFECTS OF DDT ON A MODEL ECOSYSTEM

A model ecosystem is used here to illustrate the effects of DDT waste product disposal at U.S. manufacturing sites operated from the mid-1940's to the late 1960's. Data that would accurately and completely define the extent of the hazards resulting from DDT contamination at particular sites are not available. Thus, a hypothetical site was created to demonstrate an approach for relating toxicological and ecological data to levels of contamination and to demonstrate the types of data required for establishing such a relation. The model site was developed from limited data available from actual contaminated sites[170-184] and from hypothetical circumstances (such as geology and hydrology) offered for the purpose of demonstration. The following topics are considered: manufacturing practices, composite hypothetical site, observed DDT concentrations, predicted effects, and decontamination objectives.

Manufacturing Practices

The contaminated areas of primary concern are those in the vicinity of sites previously used for the manufacture of DDT, typically following World War II until the late 1960's. As a result of manufacturing, handling, and disposal practices prevalent then, large quantities of DDT and its isomers and analogs were conveyed by surface water runoff through drainage ways into traversing streams that empty into lakes and major rivers. Depending on the manufacturing site, the methods of DDT handling and storage, and the time manufacturing ceased, there are wide ranges of possible levels of site contamination. During the manufacturing period, it is possible that tons of DDT in the form of blocks were present on the ground surface, readily accessible to leaching. After the plants were closed, massive quantities of DDT were either disposed of in burial sites and landfills, destroyed by incineration, or simply left on the ground surface.

The DDT residues in areas surrounding manufacturing sites built up over the years as process water containing DDT was discharged to settling ponds or ditches. Analyses of soil, sediment, water, and biological samples showed that undegraded DDT at some sites was being leached to surrounding areas. For example, fish caught in a major river about one mile from a contaminated site contained as much as 500 ppm DDT, two orders of magnitude greater than the maximum concentration allowable for interstate commerce. Biological surveys of the streams in contaminated areas indicated that species diversity is adversely affected in these areas.[185]

Due to DDT's low solubility in water, the most highly contaminated areas other than DDT storage or burial sites are streambed sludges. This is because the waterways leading from a plant act primarily as carriers for suspended DDT, which settles out in the streambeds. In addition, areas that are not vegetated pose a particular problem since erosion by wind and rain can carry land contaminants into surface waters. Similarly, open dumps of waste DDT can be constantly eroded by surface runoff.

Composite Hypothetical Site

A map of a composite hypothetical site is given in Fig. VII-1. In later sections of this report, the effects (approximate) of the site configuration on the environmental impacts of various DDT concentrations are considered.

The features of the composite hypothetical site are:

1. the DDT manufacturing site discharging to a large drainage ditch
2. a train of shallow lakes and wetlands containing food fish and surrounded by natural areas
3. spring flooding, periodically causing redistribution of sediments
4. ultimate drainage of DDT-containing waters into the river, which is open to boating and fishing
5. the possibility of free movement of fish and other wildlife from lakes to and from the river.

Thus, there are wetland areas where DDT in sediments can persist over many years. There are also physical and biological mechanisms for the periodic redistribution of DDT in the environment. Finally, DDT can enter wildlife and human food chains in many ways.

Observed DDT Concentrations

Concentrations of DDT in soil, sediment, and various water bodies as well as in various wildlife species are listed in Table VII-1 as a function of the downstream distance from the DDT plant. For simplification, it is assumed that the waste DDT that is buried or landfilled is located at distance zero and, because the principal carrier of DDT is water, that concentrations of DDT in water and underlying sediment are indicative of the level of contamination at each downstream distance. (The referenced data are those for actual areas surrounding DDT plants. Some of these data, however, correspond to samples collected and analyzed more than 15 years ago and, thus, may not be representative of present conditions in the areas. These data, possibly out of date, are included

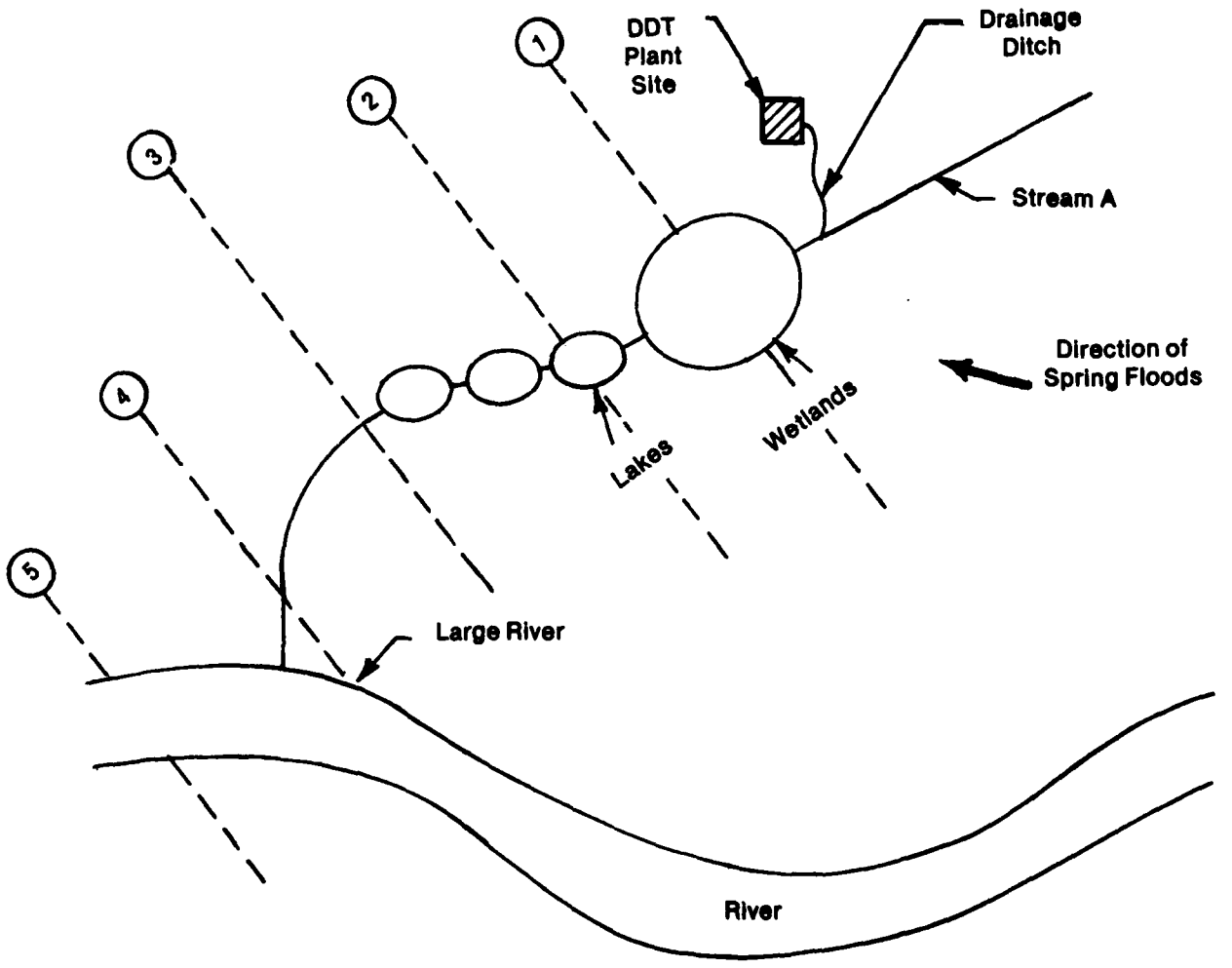


Fig. VII-1. Map of Model DDT Plant Site

TABLE VII-1. AREA AND WILDLIFE CONTAMINATION LEVELS FOR VARIOUS SITES

Approximate Distance from Plant Site (miles)	Approximate Location	Medium Sampled in Area	Reference	Concentration of DDT in Area[a]	Wildlife Species Sampled	Reference	Concentration of DDT[b] (ppm) Muscle	Fat
0	Ground surface of abandoned production area	Open dumps of waste DDT, 100 lb		100%				
0	Basement of abandoned buildings	Blocks of DDT covered with clay, 3 tons		100%				
0	Domestic sewer in plant	Water	181	6.0 ppb				
0	Drainage ditch	Water	181	2.7 ppb				
0-0.5	Drainage ditch	Sediment	181	70,000 ppm				
0.5	Closed drainage ditch	Topsoil	181	110 ppm				
1	Closed drainage ditch	Topsoil	181	0.1 ppm				
1	Stream A	Water	181	2.3 ppb	Crow	179	40	750
		Sediment		1,000 ppm	Rabbit	179	1	17
					Opossum	179	23	240
					Fox	179	27	50
					Fish	183	314	
					Fish	184	20-200	800-2,817
					Birds	184	10	
					Fish	182	300	
					Deer	183	0.2	
2	Lake	Water		1 ppb	Birds (herbivores)		2	50
		Sediment		100 ppm	Birds (carnivores)		20	500
					Fish		200	1,000

TABLE VII-1. (Cont.)

Approximate Distance from Plant Site (miles)	Approximate Location	Medium Sampled in Area	Reference	Concentration of DDT in Area[a]	Wildlife Species Sampled	Reference	Concentration of DDT[b] (ppm) Muscle	Fat
					Mammals		5	50
3	Lake	Water Sediment	180	0.5 ppb 6 ppm				
3	Bayou	Water Sediment	180	0.5 ppb 15 ppm				
4	Creek[c]	Water Sediment	--	0.1 ppb 5 ppm				
5	River	Water Sediment	181	0.03 ppb 1 ppm	Shad	182	71	
					Carp	182	29	
					Bass	182	6	
					Catfish	184	122	
					Bass	184	112	
					Sunfish	184	7	
					Bluegill	184	35	
					Fish	178	412	
					Birds (herbivores)		0.05	1
					Birds (carnivores)		1.0	10
					Mammals		0.5	5

a. DDT solubility in water = 1.2 ppb.
b. FDA tolerance for fish = 5 ppm.
c. Hypothetical data.

here to demonstrate an approach for relating toxicological and ecological effects to levels of DDT contamination.)

The concentrations of DDT in wildlife at various distances downstream from the hypothetical site are listed in Table VII-2, along with the concentrations of DDT in water bodies and sediments at these distances. It can be seen that the high concentrations in water and sediment at the shorter distances are reflected in high concentrations in the tissues of the species sampled. Conversely, the concentrations at a distance of 5 miles approach the average levels in the United States. For these data, the differences between concentrations found in muscle and fat were estimated from actual measurements.

Predicted Effects

The ultimate objective of this analysis is to predict potential site-specific environmental impacts of DDT contamination. The predictions, in their simplest form, relate environmental impacts to concentrations of DDT in soil and water. With such information and analyses of DDT in soil (sediment) and water samples, one can estimate impacts of environmental contamination and the benefits of cleaning up the soil and water to known levels. Preceding sections of this report provide evidence that currently available literature data are sufficient to relate environmental impacts to four types of exposure information: DDT concentrations in an affected organism, dietary DDT levels, acute or chronic doses of DDT, and the DDT concentration in water (for aquatic species). If these four types of information can be related to soil and water concentration data, the objective will be met.

USAMBRDL has devised a procedure for estimating safe exposure levels, called preliminary pollutant limit values (PPLVs), from laboratory or field data to protect the health of humans and other animals.[5,187] This procedure assumes an equilibrium (or steady state) relationship for a pollutant distributed among soil or sediment, water, and biota. However, as is evident in Tables VII-1 and VII-2, sediment:water and fish:water ratios vary with distance from the model site. Apparently, the PPLV algebra fails for DDT concentrations that approach the water solubility limit. Thus, an alternative procedure is required to relate health effects to environmental contaminant levels. For the model site, field data on concentrations of DDT in soil, water, and biota are adequate to predict health and environmental effects in qualitative terms.

Data presented earlier on the toxicological effects of DDT on wildlife are summarized in Fig. VII-2 and Table VII-3. Predicted impacts of DDT contamination at the model site are summarized in Table VII-4, which was derived from data presented in Tables VII-2 and VII-3. Acute toxicity is predicted to be a problem for predatory and fish-eating birds, sensitive fish species, and sensitive amphibian species at distances up to 2 miles from the DDT plant. Very sensitive fish species might be affected over the next few miles. No animal species are predicted to suffer acute toxicity symptoms at greater distances.

TABLE VII-2. ENVIRONMENTAL CONCENTRATIONS OF DDT AT THE MODEL SITE (ppm)

Sample	Reference	Distance from Plant Site (miles) 1	2	5	Average U.S. Levels	Reference
Water	181	0.0023	0.001	0.00003	0.000008-0.000144	16
Sediment		1,000(440)[a]	100(100)	1(33)	0.17	
Muscle Tissue						
Birds	179,184	25(11)				
Predatory and fish-eating			2(2)	0.05(1.7)		
Non-predatory			20(20)	1(33)		
Fish	182-184,186	300(130)	200(200)	100(3,300)		
Mammals	179,184	13(5.7)	5(5)	0.5(17)		
Fat						
Birds	179,184	750(330)				
Predatory and fish eating			50(50)	1(33)		
Non-predatory			500(500)	10(330)		
Fish	182-184,186	1,800(780)	1,000(1,000)	500(16,000)		
Mammals	179,184	130(57)	50(50)	5(170)	2.3	16

a. Values in parentheses are non-steady state concentration factors times 10^{-3}.

TABLE VII-3. TOXICOLOGICAL EFFECTS OF DDT ON TYPICAL WILDLIFE AS A FUNCTION OF CONCENTRATIONS OF DDT IN WATER, THE DIET, AND TISSUES

Medium	Approximate Concentration	Species	Effect
Water	0.01 ppb	Fish	Lethal to sac-fry
	1-20 ppb	Fish	Lethal
	5 ppb	Amphibians	Lethal
Diet	0.15 ppm	Birds	Eggshell thinning
	2 ppm	Mammals	Possible carcinogenic effects
	600 ppm	Birds	Lethal
	200 mg/kg	Mammals	Lethal
Tissues	1-5 ppm (eggs)	Fish	Lethal to sac-fry
	1 ppm (eggs)	Birds	Eggshell thinning

Reproductive failure is expected for predatory and fish-eating birds and for fish at distances up to 5 miles and more from the DDT plant site. Repopulation of the model site with these species is to be expected only for those species with some accessible breeding populations at distances sufficient to avoid DDT-associated reproductive failure. In other words, fish and predatory birds may be found at the model site, but it is unlikely that sensitive species hatched within 5 miles of the DDT plant.

Mammals are generally much more resistant to DDT than birds or fishes. Even so, it is not improbable that fish-eating mammals, e.g., otters, could ingest toxic quantities of DDT, considering the high dietary levels (Table VII-1). Their intake might, for example, exceed the 20 ppm DDT reported to cause teratogenic or embryotoxic effects in mice. Lower levels could conceivably induce cancer, but this would not be ecologically significant because cancer from a weak carcinogen, such as DDT, would be expected to afflict only senescent individuals. Fish taken for human food within 5 miles of the site are virtually certain to exceed the 5 mg/kg limit established by the Food and Drug Administration. For average daily consumption of 18.7 g of such fish, the associated lifetime cancer risk, by the EPA's method,[28] exceeds 1 in 1,000. For consumption of fish containing 50 mg DDT/kg, the associated risk would exceed 1 in 100. (Note, however, that EPA considers the cancer risk from DDT ingestion derived from epidemiological data to represent an upper bound. The actual risk may be substantially lower.)

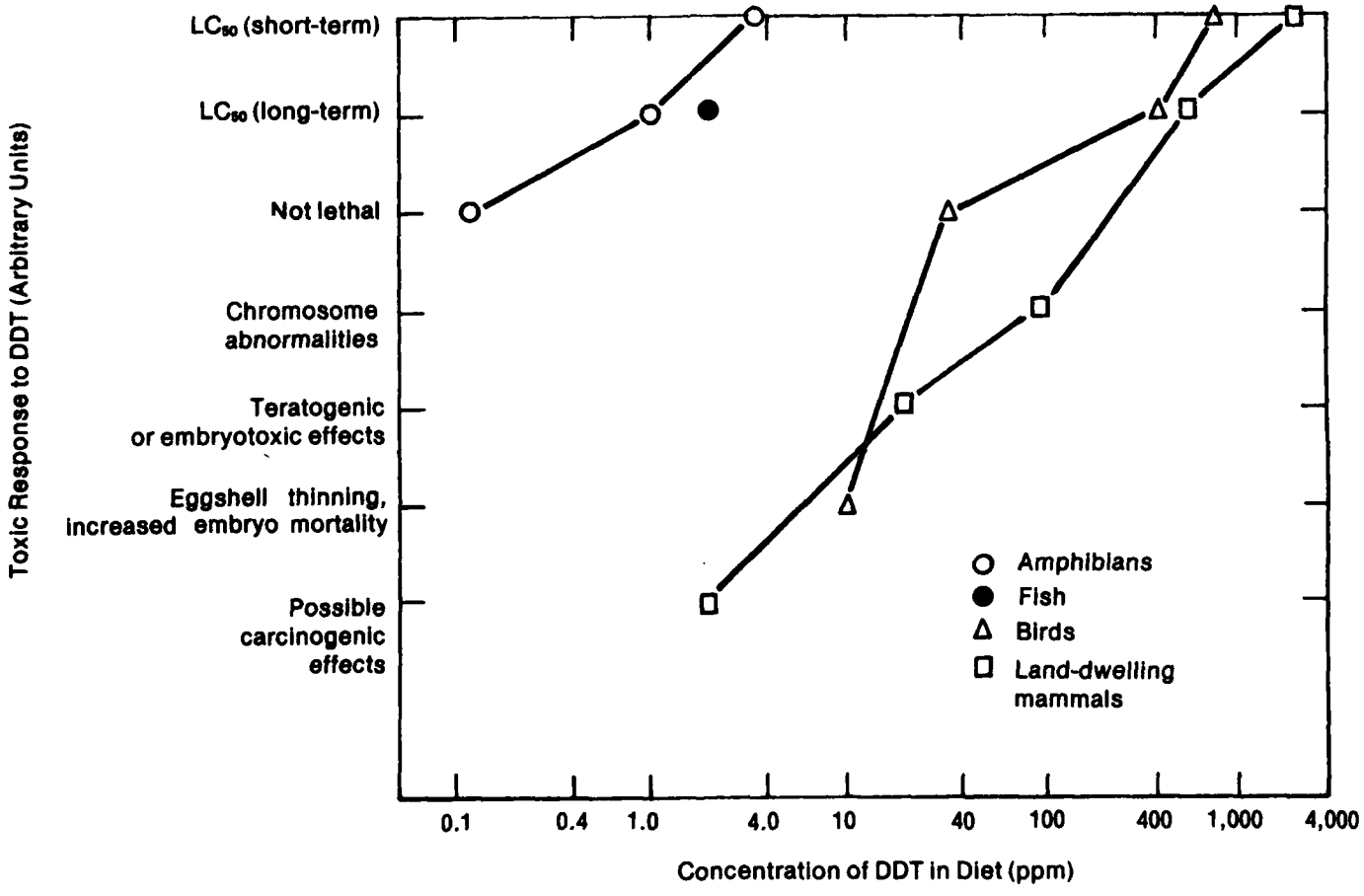


Fig. VII-2. Biological Effects of Dietary DDT Typical Receptors

TABLE VII-4. PREDICTED EFFECTS ON WILDLIFE

Effect	Distance from DDT Plant Site (miles)		
	0-2	2-5	5
Acute toxicity for	Predatory and fish-eating birds, sensitive fish, sensitive amphibians	Very sensitive fish	--
Reproductive failure due to	Eggshell thinning in predatory and fish-eating birds, death of fish sac-fry	Eggshell thinning in predatory and fish-eating birds, death of fish sac-fry	Eggshell thinning in predatory and fish-eating birds, death of fish sac-fry

Decontamination Objectives

In lieu of PPLV's, maximum environmental DDT levels for protection of wildlife have been calculated directly from model site data (Table VII-1). These are 0.1 ppb in water and 4 ppb in sediment for predatory and fish-eating birds and 0.05 ppb in water and 2 ppb in sediment for fish.

The critical effect for birds is eggshell thinning leading to reproductive failure. Available data suggest that for sensitive birds, such as the brown pelican, DDT concentrations greater than 1 ppm in the egg can cause a significant decline in reproductive success. Other studies suggest that DDT levels in bird fat are approximately 40 times the levels in bird eggs. Table VII-2 shows that the ratio of DDT in fat of predatory birds to DDT in water is approximately constant and falls in the range of 300,000 to 500,000. Assuming a ratio of 400,000, it is calculated that a DDT level in water not to exceed 0.1 ppb will provide a safe limit of 40 ppm in fat and 1 ppm in eggs. This corresponds to a sediment concentration of about 4 ppb. For less sensitive species, safe concentration limits will be higher. (It should be emphasized that these calculations assume that measured DDT concentrations are equilibrium or steady-state levels. If not, derived values could be in error by an order of magnitude or greater.)

For fish, the critical effect is mortality of sac-fry, which can occur at DDT concentrations of about 1 ppm in fish eggs (Table V-4) and estimated corresponding concentrations of 0.01 ppb in water and 0.4 ppb in sediment (assuming a sediment-to-water ratio of 40). Data of Table VII-1 indicate that reproductive success could be expected at distances greater than 5 miles from the model manufacturing site.

To establish engineering goals for cleanup efforts, benefits to wildlife and humans are predicted to occur for any degree of cleanup from present levels down to 2 ppb in sediments (0.05 ppb in water). These concentrations are so low, and the area of dispersal so great, that it may be better to focus on cleanup efforts giving the greatest reduction of total mass of DDT, accepting the fact that decreases to ppb levels in sediment will have to come through biodegradation. Regular monitoring of DDT levels in fish and waterfowl will provide a measure of restoration.

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